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Published in:
Arthritis Research and Therapy

DOI:
[10.1186/ar4576](https://doi.org/10.1186/ar4576)

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Document Version
Publisher's PDF, also known as Version of record

Publication date:
2014

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Dolff, S., Abdulahad, W. H., & Kallenberg, C. G. M. (2014). Response to 'T-helper 17 cell cytokines and interferon type I: partners in crime in systemic lupus erythematosus?'. *Arthritis Research and Therapy*, 16(3), [409]. <https://doi.org/10.1186/ar4576>

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LETTER

Response to 'T-helper 17 cell cytokines and interferon type I: partners in crime in systemic lupus erythematosus?'

Sebastian Dolff^{1*}, Wayel H Abdulahad² and Cees GM Kallenberg²

See related research by Brkic et al., <http://arthritis-research.com/content/16/2/R62> and related letter by Brkic et al., <http://arthritis-research.com/content/16/3/410>

We read with great interest the article by Brkic and colleagues in a recent issue of *Arthritis Research & Therapy* [1]. In that study, the authors investigated the distribution of T helper (Th) subsets which produce IL-17A, IL-17 F, IL-21, and IL-22 in patients with systemic lupus erythematosus (SLE) in relation to their genetic IFN type I signature. Patients with an IFN type I-positive signature showed increased percentages of IL-17A- and IL-21-producing CCR6⁺ T cells. From these results, the authors conclude that IFN type I cells co-act with Th17 cytokines in the pathogenesis of SLE. Surprisingly, they excluded CD25⁺ T cells from their analysis. In a previous study, we showed that Th cells from SLE patients expressing CD25^{med} and CD25^{high} are also able to produce IFN-γ and IL-17A [2]. Therefore, it would be relevant to assess cytokine expression in CD4⁺CD25⁺ T cells from IFN type I-positive and IFN type I-negative SLE patients. Furthermore, it should be proven that the genetic signature is solely responsible for the increased IFN production by Th cells. In addition, their finding that CCR6⁺ T cells are capable of producing IL-21 indirectly confirms our previous observation that IL-17⁺ T cells are a main source of IL-21 in patients with SLE [3]. Possibly, IL-21 is orchestrating the Th1/Th17 axis.

Finally, we agree that there might be a co-activity between IFN-I- and IL-17-producing cells as described by Brkic and colleagues. However, considering our findings that T cells with a regulatory phenotype are able to produce IFN-γ and IL-17A in patients with SLE, we suggest that primarily the plasticity of T cells is altered in patients with SLE [4].

Abbreviations

IFN: Interferon; IL: Interleukin; SLE: Systemic lupus erythematosus; Th: T helper.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

All authors contributed to the interpretation of data. SD drafted the manuscript. All authors read and approved the final manuscript.

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Published: 09 Jun 2014

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10.1186/ar4576

Cite this article as: Dolff et al.: Response to 'T-helper 17 cell cytokines and interferon type I: partners in crime in systemic lupus erythematosus?'. *Arthritis Research & Therapy* 2014, **16**:409

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